Homosexuality roots: Precocious puberty?

Homosexuality has over the years been linked to everything from a domineering mother to attendance at boarding school to the absence of a gag reflex. None of these explanations of homosexual origins has withstood scientific evaluation. The most recent theory — that homosexual preferences are learned during puberty — is now undergoing scientific scrutiny and, as two recent studies show, the preliminary evidence is mixed.

Social psychologist Michael D. Storms of the University of Kansas last year introduced a theory of homosexuality that holds that the timing of sexual maturation is the key determinant of homosexual or heterosexual responsiveness. According to Storms's theory, children eroticize whatever psychological cues are available to them when their sex drive first develops. Children who mature prematurely — when they are still in social groups of the same sex — learn from an environment rich in homosexual cues; children who mature late, when boys and girls are more integrated socially, tend to eroticize heterosexual cues and to develop an ultimate preference for heterosexual relationships. Speaking at the annual meeting of the American Psychological Association last week in Washington, D.C., Storms presented the first data to support his controversial theory.

Storms questioned 97 male and 62 female college students about the frequency and intensity of their sexual fantasies involving both the same sex and opposite sex; he also asked the subjects to recall, using various measures, the age at which they matured sexually. The data for the male subjects, Storms reported, fit well with the theoretical model: sexually precocious children were more apt to have a "homoerotic" orientation as adults, while late maturers were apt to be more heterosexual in their preferences.

Storms's theory and preliminary findings diverge from the conclusions of last year's report from the Alfred C. Kinsey Institute for Sex Research. That report, based on interviews with nearly 1,400 subjects, had indicated that childhood sexual feelings — already crystallized by the time of puberty and unrelated to any clear social or psychological roots — strongly predicted adult homosexuality. The report strongly hinted at the possibility of biological origins for homosexuality. Indiana University sociologist Sue Kiefer Hammersmith, one of the authors, reported last week that a reanalysis of the Kinsey data did not lend support to Storms's ideas.

According to Hammersmith, an analysis of the Kinsey data indicates no significant differences in age of sexual maturity for homosexuals and heterosexuals — in contrast to Storms's contention that homosexuals mature two to three years earlier. She also reported finding no meaningful correlations between timing of sexual drive and erotic interests during adulthood. Finally, Hammersmith noted, her data indicate that homosexual men and women tended to have more — not less — social interaction with the opposite sex before high school, though such social bonding appears unrelated to adult sexual preferences.

Storms, in reply, argued that all previous research on homosexuality — including the Kinsey study — has been confused by its methods. By attempting to study sexual behavior and lifestyle in subjects who are self-identified homosexuals, he said, researchers have introduced any number of confounding variables. Data from such research cannot be used to properly test his model, which focuses only on erotic responsiveness.

Storms's model was also criticized by Johns Hopkins University biologist James D. Weinrich, co-editor of the new book *Homosexuality* (Sage, 1982); Weinrich said that the theory failed to explain childhood sissiness, which has been shown to predict adult homosexuality. According to Storms, sissiness has nothing to do with adult sexual responsiveness, but as an indicator of non-conformity, it might predict openness about one's erotic preferences.

—W. Herbert

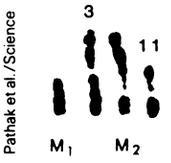
Inherited cancer genes: More evidence

Some human cancers are due to inherited genetic quirks, according to recent chromosomal and pedigree studies (SN: 5/9/81, p. 297). Now more evidence that this is the case is reported in the Sept. 3 SCIENCE by Sen Pathak of the M.D. Anderson Hospital and Tumor Institute in Houston and colleagues.

In fact, in the opinion of the researchers, "This finding adds further support to the existence of specific human cancer genes." And in the opinion of Andrew J. Cohen of the University of Massachusetts Medical Center in Worcester, who has done related research, "it confirms that there may be a cancer gene on chromosome number 3."

Pathak and his colleagues studied a 32-year-old patient with kidney cancer. First they examined death certificates and medical records from his family and found cases of kidney cancer over three generations, implying that the patient's kidney cancer was inherited. Then they took 50 kidney cancer cells from the patient and made a karyotype of the chromosomes in each cell. (A karyotype is a systematic pictorial arrangement of dividing chromosomes in a cell and in a normal human karyotype there are 23 pairs of chromosomes.) They stained the chromosomes with a special dye so they could easily identify the structure of each chromosome. They analyzed the chromosomes in the 30 karyotypes that had stained the best to see whether they could identify any specific chromosomal abnormalities that could be implicated in the patient's kidney cancer.

Twenty-two of the 30 karyotypes revealed one normal chromosome number 3 and one normal chromosome number 11. The other chromosome number 3 and the other chromosome number 11 in each of the 22 karyotypes had swapped genetic material. Part of the short arm of chromosome number 3 had attached to the short arm of chromosome number 11, thus leaving an excessively short number 3 and an excessively long number 11. In each of the other eight karyotypes, there were two normal copies of chromosome number 3 and two normal copies of chromosome number 11. Because three-fourths of the karyotypes contained a similar translocation between a chromosome number 3 and a chromosome number 11, the patient's kidney cancer may well have resulted from an inherited chromosome 3-chromosome 11 translocation, the researchers conclude.



Pathak et al./Science

A cancerous kidney cell contains one normal chromosome number 3 and one normal chromosome number 11 as well as a smaller chromosome number 3 (M_1) and a larger chromosome number 11 (M_2).

However, they concede, it is possible that the patient's cancer wasn't due to the translocation per se but rather to a deletion of a critical gene as genetic material was transferred from chromosome number 3 to chromosome number 11. If so, they point out, kidney cancer would join retinoblastoma and Wilms' tumor as examples of an inherited human cancer caused by the deletion of a gene (SN: 5/9/81, p. 297).

Cohen is also inclined to think that the patient's cancer may have been due to a gene deletion rather than to a translocation per se. On the other hand, he points out, the chromosomal translocation that he and his colleagues identified in eight family members with kidney cancer also involved chromosome number 3 (they found translocation to chromosome number 8). Thus it's possible, Cohen conjectures, that the translocation that Pathak and his team identified, as well as the one that he and his colleagues identified, may have activated some gene on chromosome number 3. And this gene, he says, might have been the cancer trigger.

—J.A. Treichel